U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 22 July 2004

CASE NO.: 2003-BLA-255

In the Matter of:

HELEN M. MUCKER, survivor of
GEORGE MUCKER
Claimant

v.

BETHENERGY MINES, INC.
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party in Interest

APPEARANCES:

John D. Gibson, Esq.
For the Claimant

John J. Bagnato, Esq.
For the Employer

Before: DANIEL L. LELAND
Administrative Law Judge

DECISION AND ORDER - DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. § 901 *et seq*. In accordance with the Act and the pertinent regulations, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as black lung.

A formal hearing was held in Ebensburg, Pennsylvania on May 5, 2004, at which all parties were afforded full opportunity to present evidence and argument, as provided in the Act and the regulations found in Title 20 Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title. At the hearing, Director's exhibits (DX) 1-66 and Employer's exhibits (EX) 1-3 were admitted into evidence. Claimant and Employer submitted closing briefs.

ISSUE

The only issue is the cause of the miner's death.

FINDINGS OF FACT AND CONCLUSIONS OF LAW[1]

Procedural History

Helen M. Mucker (Claimant) is the surviving spouse of George Mucker (miner), who was born on December 28, 1924 and died on July 22, 2000. (DX 1). Claimant filed a claim for survivor's benefits on August 10, 2000. (DX 1). The district director denied benefits on May 22, 2001, which Claimant appealed on June 19, 2001. (DX 27, 28). The case was referred to the Office of Administrative Law Judges (OALJ) on August 13, 2001, and a formal hearing was held before Administrative Law Judge Michael P. Lesniak on January 23, 2002. (DX 32, 39). On June 20, 2002, Judge Lesniak issued a Decision and Order – Remanding Claim to the district director for further development of the record. (DX 41). Specifically, the district director was ordered to obtain a supplemental report from Dr. Perper in which he reviews all of the evidence in the record and a supplemental report from Dr. Mittal as to whether the autopsy slides were representative of the lung tissue samples. Employer was ordered to submit Dr. Awan's report, hospitalization records from 1989, and a complete copy of Dr. Bush's report. Claimant was ordered to submit a statement regarding the miner's use of inhalers. After this additional evidence was developed and/or submitted to the district director, he denied benefits on April 18, 2003.[2] (DX 58). Claimant appealed on May 7, 2003, and the case was referred to the OALJ on July 29, 2003. (DX 60, 65).

At the hearing, Employer stipulated that the miner had thirty-five years of coal mine employment. (TR 6). Previously, Employer has stipulated that the miner had simple pneumoconiosis which arose from his coal mine employment. (DX 41, p. 2). Claimant testified that the miner used inhalers because he could not breathe. Claimant explained that the miner would choke on mucous in his throat, and he would spray the inhaler in his throat to get the mucous out. (TR 12-13). Claimant testified that the miner had this problem every day of the last three or four years of his life. (TR 13-14).

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[1] The following abbreviation has been used in this decision and order: TR = transcript of hearing.

[2] In his Proposed Decision and Order, the district director erroneously referred to this claim as a request for modification. (DX 58). Neither party had requested a modification of the district director's decision, but rather this claim was before the district director because of a remand by Judge Lesniak for the development of additional evidence. Therefore, § 725.310 is not applicable to this claim.

Medical Evidence

The summary of medical evidence in the Decision and Order – Remanding Claim issued by Judge Lesniak on June 20, 2002 is incorporated by reference. The following summary is of evidence submitted since the issuance of that decision and order.

Medical Reports

Dr. Rashid A. Awan drafted a letter on May 14, 1990. (DX 45). Dr. Awan had treated the miner for "the past several years" for chronic obstructive pulmonary disease and status post coronary artery bypass surgery. Dr. Awan stated that the miner's chronic obstructive pulmonary disease was stable with medication (he was prescribed an inhaler), but that he became short of breath walking less than one block. Dr. Awan concluded that the chronic obstructive pulmonary disease was "probably" secondary to his coal mine employment, even though the miner's last office chest x-ray, dated November 24, 1986, was normal and Dr. Awan had not reviewed any of his pulmonary function tests.

Dr. Stephen T. Bush, an anatomic and clinical pathologist, reviewed the medical records and drafted a medical report dated January 30, 2001. (DX 21, 45). Dr. Bush noted that the coal worker micronodules measured up to 0.4 cm and were surrounded by focal dust emphysema. Dr. Bush stated that there was an average of five coal worker lesions per slide, and that approximately five percent of the lung tissue was destroyed by the lesions. Dr. Bush concluded that the miner had mild to moderate simple coal workers' pneumoconiosis. Dr. Bush noted a moderate number of pulmonary macrophages containing iron pigment, which is indicative of chronic congestive heart failure. Dr. Bush also noted a mild degree of centrilobular emphysema independent of any dust pigment deposition. Dr. Bush attributed the centrilobular emphysema to the miner's history of cigarette smoking. Dr. Bush opined that the miner's death resulted from severe cardiac disease due to coronary atherosclerosis. Dr. Bush's opinion is based on: (1) the miner's severe coronary artery atherosclerosis with temporary relief of obstruction upon surgery, (2) history of systemic hypertension aggravating the effects of coronary artery disease in the medical records, (3) the miner's heart weighed 1200 grams at autopsy, (4) evidence of biventricular hypertrophy, and (5) evidence of congestive heart failure (prominent bilateral pleural effusions, severe chronic passive congestion of the liver with centrilobular ischemic change, and iron pigment in the lungs). Dr. Bush found that the coal workers' pneumoconiosis was too limited in degree and extent to have contributed to or caused the miner's death. Finally, Dr. Bush concluded that the miner did not have cor pulmonale because the clinical records and autopsy findings revealed biventricular hypertrophy consistent with a chronically failing left ventricle that secondarily affected the right ventricle, causing both ventricles to be thickened. Dr. Bush stated that "[i]f primary lung disease were to cause thickening of the right ventricle, the disease would necessarily be severe, diffuse, bilateral changes that cause obstruction of pulmonary vessels and an increase in pulmonary artery pressure. Such disease is not present in the lungs clinically, radiologically or pathologically." (DX 21, p. 4; DX 45, p. 4).

Dr. Vimal Mittal drafted a supplemental letter dated August 8, 2002. (DX 44). Dr. Mittal stated that the lung slides were representative of coal workers' pneumoconiosis. Also, he

stated that the slides revealed micronodules and macules involving sixty percent of the lung surface.

Dr. Larry E. Hurwitz drafted a supplemental report dated September 12, 2002. (DX 47). Dr. Hurwitz stated that the miner's long-term use of inhalants did not change his opinion that coal workers' pneumoconiosis did not contribute to the miner's death. Dr. Hurwitz stated that "[the miner] had long-standing coronary disease from 1976. He had symptomatic myocardial ischemia following myocardial infarction. Bronchodilating medication has classically been used for cardiovascular purposes in patients with left ventricular dysfunction." (DX 47, p. 2).

After reviewing all of the medical evidence in the record, Dr. Joshua A. Perper drafted a supplemental report dated December 23, 2002. (DX 50). Dr. Perper offered criticisms of several physicians' opinions. First, Dr. Perper criticized Dr. Bush's report because he did not diagnose cor pulmonale. Dr. Perper stated that the presence of coronary arteriosclerotic heart disease and associated ventricular hypertrophy does not preclude the presence of concurrent lung disease and related cor pulmonale. Also, Dr. Perper explained that "the relative percentual increase in [right ventricle] thickening may be a reliable marker as to whether it was or not a manifestation of cor pulmonale," and that the miner had one hundred percent increase in thickening. Further, he found that the miner had mild to moderate sclerosis of small intrapulmonary blood vessels upon microscopic examination, another indicator of pulmonary hypertension and cor pulmonale. (DX 50, p. 14). Thus, Dr. Perper concluded that the miner had cor pulmonale. Second, Dr. Perper criticized Dr. Fino's reports because there was evidence of a respiratory impairment in the medical records, including positive chest x-ray interpretations, low normal or abnormally low pulmonary function tests, evidence of chronic hypoxemia, the miner's treatment with bronchodilators and at times oxygen, and repeated diagnoses of chronic obstructive pulmonary disease. Dr. Perper also criticized Dr. Fino's reports because his tables of pulmonary function studies revealed chronic hypoxemia and consistently low MVV values, yet Dr. Fino invalidated every MVV value. Dr. Perper stated that Dr. Fino's citations to the medical literature are outdated, and that the current literature reveals that individuals with chronic obstructive pulmonary disease have a greater incidence of cardiac arrhythmia. Third, Dr. Perper criticized Dr. Hurwitz's report because his denial of pulmonary co-morbidity is "in clear disagreement" with the medical evidence revealing subjective and objective findings of lung disease and pulmonary/hypoxemic dysfunction. Dr. Perper also stated that Dr. Hurwitz's dismissal of the laboratory findings of pulmonary hypertension as secondary to left heart failure is improper because some of the measurements were taken when the miner was not in left heart failure and the findings of right ventricular hypertrophy and intrapulmonary vascular changes are consistent with pulmonary hypertension. Finally, Dr. Perper stated that bronchodilators are not standard medications in the treatment of left ventricular failure, criticizing Dr. Hurwitz's comment that bronchodilating medication has classically been used for cardio-vascular purposes.

Dr. Perper concluded that the miner had chronic pulmonary disease based on: increased anterior diameter of the chest, reduced breathing sounds and muffled heart sounds, several chest x-rays that were interpreted as positive for pneumoconiosis, the pulmonary function studies revealed mild to marked respiratory impairment, the arterial blood gas tests revealed hypoxemia, and the medical records revealed that the miner was prescribed bronchodilators for a respiratory condition. Dr. Perper also concluded that the miner had cor pulmonale based on: the objective

cardiac tests revealed dilatation of the right ventricle and evidence of pulmonary hypertension, the gross autopsy findings revealed marked right ventricular hypertrophy, and the microscopic autopsy findings revealed mild to moderate sclerosis of the intrapulmonary blood vessels. Dr. Perper stated that the miner's severe arteriosclerotic cardiovascular disease was not caused by chronic obstructive pulmonary disease, coal workers' pneumoconiosis, or coal dust exposure, but that these conditions adversely affected his severe arteriosclerotic and hypertensive cardiovascular disease. Thus, Dr. Perper opined that simple coal workers' pneumoconiosis was a contributory cause and hastening factor of the miner's death "both directly and through hypoxemia precipitating a fatal arrhythmia on the background of the severe coronary heart disease." (DX 50, p. 30). Dr. Perper explained that he changed his opinion based on the voluminous medical records that he had reviewed.

Dr. Hurwitz drafted a supplemental report dated January 27, 2003. (DX 55). Dr. Hurwitz disagreed with Dr. Perper's opinion. First, Dr. Hurwitz stated that the records from the July 10, 2000 cardiac catheterization revealed that the miner's oxygen saturation on room air was normal, demonstrating that he did not have chronic hypoxia. Second, Dr. Hurwitz stated that Dr. Perper erred in stating that pulmonary hypertension only occurs when an individual is in overt congestive heart failure. Dr. Hurwitz explained that pulmonary hypertension secondary to left ventricular dysfunction produces the same pathophysiologic changes in the right heart as an individual with pulmonary hypertension secondary to intrinsic lung disease. Dr. Hurwitz stated that the miner's persistent left ventricular dysfunction with or without overt decompensation is the principal factor related to his pulmonary hypertension, and thus a diagnosis of cor pulmonale cannot be made. Finally, Dr. Hurwitz clarified that his comments regarding the use of bronchodilators for cardiovascular purposes was not "intended to imply that my understanding of the use of the medication in [this] case was specifically for the treatment of his left ventricular dysfunction." (DX 55, p. 2).

Dr. Bush drafted a supplemental report dated February 3, 2003. (DX 56). Dr. Bush disagreed with Dr. Perper's report. Dr. Bush noted that the miner's seven hospitalizations between 1994 and 2000 were due to severe cardiac disease, not lung disease. He also noted that during the last hospitalization, the physicians did not describe the miner as hypoxic and the arterial blood gas tests were normal. The miner did become short of breath during the cardiac catheterization on July 10, 2000, but that was due to the insertion of the catheter into the left main coronary artery. Dr. Bush noted that that there was no evidence of hypoxemia either before or after the angiography. Dr. Bush disagreed with Dr. Perper's characterization of arrhythmia as a mechanism of the miner's death. He noted that the miner had cardiac arrhythmia for many years before his death, and that cardiac disease due to coronary atherosclerosis predisposed the miner to cardiac arrhythmia without hypoxemia. Dr. Bush reiterated his opinion that the miner did not have cor pulmonale and cited medical literature that a diagnosis of cor pulmonale is not appropriate when there is left and right ventricular thickening, especially when the left ventricular thickening is due to diseases that primarily affect the left side of the heart. The miner had systemic hypertension, severe coronary artery disease, myocardial infarctions, and repeated episodes of congestive heart failure, all of which are conditions that affect the left side of the heart. Moreover, Dr. Bush stated that Dr. Perper's reference to the arithmetic unidimensional measure of ventricular thickness is a "profoundly" inaccurate method for assessing ventricular hypertrophy. Finally, Dr. Bush criticized Dr. Perper's diagnosis of pulmonary hypertension. Dr.

Bush stated that Dr. Perper's observation of mild to moderate sclerosis of the small intra-pulmonary blood vessels is "silly" because conventional staining methods turn the various tissues pink and indistinguishable from one another. Dr. Bush also explained that pulmonary hypertension does not develop unless a severe pulmonary disease is present, and there is no evidence microscopically, radiologically, or clinically that the miner suffered from a severe lung disease, and thus a diagnosis of pulmonary hypertension is inappropriate.

Dr. Fino drafted a supplemental report dated February 6, 2003. (DX 57). First, Dr. Fino stated that the pulmonary function study tables in his December 7, 2001 report do not reveal evidence of chronic hypoxemia, contrary to Dr. Perper's opinion. Dr. Fino stated that every room air arterial blood gas test performed between 1979 and 1989 was normal without hypoxemia. Also, he stated that the eleven arterial oxygen saturation measurements taken during the July 10, 2000 cardiac catheterization were normal. Further, Dr. Fino stated that there is no evidence in the medical records that the miner had hypoxemia. Second, Dr. Fino stated that the miner's pulmonary function studies were normal when he gave good effort, and that when he did not give good effort, the studies are invalid and "should not be mistaken for being abnormal." (DX 57, p. 3). Also, Dr. Fino stated that all of the MVV values are invalid, but when compared to the DOL standards, all of the MVV values are normal. Thus, Dr. Fino concluded that the medical records do not support Dr. Perper's finding of hypoxemia, abnormal pulmonary function studies, or chronic obstructive pulmonary disease. Finally, Dr. Fino stated that the articles cited by Dr. Perper regarding the connection between chronic obstructive pulmonary disease and arrhythmia do not address his comments, as Dr. Fino's citation to medical studies dealt with chronic hypoxemia not predisposing an individual to cardiac arrhythmias.

Dr. Fino was deposed on December 2, 2003. (EX 1). Dr. Fino discussed the miner's shortness of breath. He explained that in order for a lung disease to produce shortness of breath, there must be valid pulmonary function studies that reveal an abnormality. Because all of the miner's pulmonary function studies were normal, Dr. Fino found that the miner's shortness of breath was not due to lung disease. (EX 1, p. 15). Dr. Fino explained that an individual's shortness of breath can also be due to lung disease when the individual has problems transferring oxygen from the air sacs in the lungs to the bloodstream. Dr. Fino testified that all of the arterial blood gas tests and diffusing capacity tests were normal, and so he concluded that there is no pulmonary reason for the miner's shortness of breath. (EX 1, p. 16). Dr. Fino testified that the miner's severe coronary artery disease would cause shortness of breath on exertion. (EX 1, p. 17). Dr. Fino testified that there is no pulmonary reason to prescribe bronchodilators for the miner since he did not suffer from an obstructive impairment. He explained that when shortness of breath is due to cardiac disease, it will be alleviated by stopping exertional activity that caused the shortness of breath. Dr. Fino testified that he has seen individuals with cardiac disease prescribed bronchodilators, not because they have a pulmonary impairment, but because the medication requires the individuals to stop their activity, which allows the heart to "catch up" on pumping blood and the shortness of breath goes away. (EX 1, pp. 19-20). Dr. Fino opined that the miner's death was due to his heart disease, and that there is no causal relationship between his death and coal dust exposure. (EX 1, p. 21). Dr. Fino disagreed with Drs. Mittal and Perper's opinion that the miner's death was due to hypoxemic arrhythmias because hypoxemic arrhythmias are caused by hypoxemia, and there is no evidence that the miner had hypoxemia. (EX 1, p. 24).

Dr. Bush was deposed on December 3, 2003. (EX 2). Dr. Bush testified that there is no evidence that hypoxemia contributed to the miner's death because there is no evidence that the miner suffered from hypoxemia. (EX 2, p. 12). Dr. Bush explained that congestive heart failure is the inability of the heart to effectively pump blood through the heart chambers, and it can lead to the development of pulmonary edema. The miner had 1500 cc's of pleural fluid in the left side of the pleural cavity and 1000 cc's of pleural fluid in the right side of the pleural cavity, which Dr. Bush characterized as a "tremendous" amount of pleural effusion from congestive heart failure. (EX 2, pp. 15-16). Dr. Bush explained that cor pulmonale is the inability of the right ventricle to effectively pump blood out of the heart chamber and is the result of a primary lung disease. In the case of coal workers' pneumoconiosis, the scarring created by the inhaled coal dust compresses the blood vessels and raised the blood pressure, causing the right side of the heart to work harder. (EX 2, pp. 16-17). Dr. Bush disagreed with Dr. Perper's diagnosis of cor pulmonale because the miner's lungs were not severely involved by a primary lung disease and there was evidence that he had a left ventricle abnormality that caused the right ventricle abnormality, thus ruling out cor pulmonale. (EX 2, pp. 17-18).

Dr. Hurwitz was deposed on January 8, 2004. (EX 3). Dr. Hurwitz testified that shortness of breath is a non-specific symptom, but in his opinion the underlying factor for the miner's shortness of breath was his longstanding coronary artery disease, angina pectoris, and progressive left ventricular dysfunction. (EX 3, pp. 11, 21). Dr. Hurwitz testified that the miner was taking Atenolol, a beta blocker, which is prescribed to patients with coronary artery disease and angina, and that one of the side effects of Atenolol is bronchial constriction. Dr. Hurwitz testified that the miner was also taking Albuterol, a beta agonist and bronchodilator, which relieves bronchial constriction. (EX 3, p. 11-12). Dr. Hurwitz stated that the medical records did not indicate why Albuterol was prescribed to the miner, but that it was prescribed after he had been prescribed beta blockers. (EX 3, pp. 20-21). Dr. Hurwitz testified that the miner did not have cor pulmonale because he had severe left-sided heart disease, congestive heart failure, and essentially no coronary circulation, but no evidence of jugular venous hypertension. (EX 3, p. 14). Dr. Hurwitz disagreed with Dr. Perper's opinion regarding hypoxemia's role in the miner's death because the clinical records do not indicate that the miner had hypoxemia. (EX 3, p. 16). Dr. Hurwitz testified that the miner had an acute ischemic event during the July 10, 2000 cardiac catheterization. Dr. Hurwitz explained that when the catheter was inserted in the miner's left main coronary artery, all of the circulation to his heart stopped and he had to be treated with intravenous nitroglycerine and oxygen. (EX 3, pp. 17-18). Dr. Hurwitz stated that "had the operator not been there, [the miner] would have died [during the catheterization]" because he had "essentially no back up." (EX 3, p. 18). Dr. Hurwitz concluded that the miner's death was due solely to his heart disease.

Hospitalization and Other Records

The miner was hospitalized at Mercy Hospital from February 23, 1989 to March 3, 1989. (DX 45). The miner underwent a triple coronary artery bypass graft on February 24, 1989. The miner underwent fifteen arterial blood gas tests from February 23-25, 1989, all of which were normal. The miner's principal diagnosis was coronary artery disease. Based on the patient history, the miner was also diagnosed with silicosis and partial lobe black lung.

Claimant drafted a letter to the district director dated August 12, 2002. (DX 46). Claimant stated that the miner began using an inhaler in January of 1985. Claimant stated that the miner used the inhaler every morning before descending the stairs and every time he walked up the stairs during the day. Claimant also stated that the miner used the inhaler when he walked outside "for any given distance" and before bed to help him sleep. Finally, Claimant stated that the miner took medication to relieve his productive cough.

The record also contains a printout from Shafer Drug Store dated October 3, 1996 to July 19, 2000, which confirms that the miner filled a prescription for Albuterol twice in 1996, three times in 1997, twice in 1998, seven times in 1999, and three times in 2000. (DX 46).

The record contains a letter from Richard H. Thompson, Director of Pennsylvania's Bureau of Workers' Compensation to Patricia Fyock (the miner and Claimant's daughter) dated September 13, 2002. (DX 48). Mr. Thompson stated that the miner was awarded partial disability benefits due to his pneumoconiosis under the Pennsylvania Workers' Compensation Act from April 14, 1983 to November 11, 1992.

Conclusions of Law

Benefits are provided to eligible survivors of a miner whose death is due to pneumoconiosis. § 718.205(a). In claims filed on or after January 1, 1982, death will be considered due to pneumoconiosis (1) where competent medical evidence establishes that the miner's death was due to pneumoconiosis, or (2) where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or (3) where the presumption in § 718.304 is applicable. § 718.205(c). Pneumoconiosis is a substantially contributing cause of death if it hastened the miner's death. § 718.205(c)(5); *see also Lukosevicz v. Director, OWCP*, 888 F.2d 1001 (3d Cir. 1989).

Drs. Mittal and Perper concluded that coal workers' pneumoconiosis was a contributory cause of the miner's death. Drs. Bush, Fino, Griffin, Hurwitz, Naeye, and Tomashefski concluded that the miner's death was due to severe coronary atherosclerosis. There are three issues raised by the physician opinion evidence: (1) whether the miner had cor pulmonale, (2) whether the miner had hypoxemia, and (3) whether coal workers' pneumoconiosis hastened the miner's death.

Five physicians discussed whether the miner had cor pulmonale at the time of his death. Dr. Perper concluded that the miner did have cor pulmonale, whereas Drs. Bush, Griffin, Hurwitz, and Tomashefski found that the miner did not have cor pulmonale. Dr. Perper stated that the following findings support a diagnosis of cor pulmonale: mild to moderate sclerosis of the intrapulmonary blood vessels, objective cardiac tests showing dilation of the right ventricle and evidence of pulmonary hypertension, and autopsy findings of marked hypertrophy of the right ventricle. Dr. Bush questioned Dr. Perper's diagnosis of sclerosis of the intrapulmonary blood vessels because conventional staining methods turn all of the tissue pink and indistinguishable. He stated that special elastic stains are necessary to make such a diagnosis. Dr. Mittal, the prosector, did not identify the staining methods used to create the autopsy slides,

but he did identify thickened medium and small vessels in sections of the lung. While Dr. Bush's comments raise doubts about the staining methods used to create the autopsy slides, I find that Dr. Mittal's identification of thickened medium and small vessels supports Dr. Perper's finding of sclerosis of the intrapulmonary blood vessels. Dr. Hurwitz questioned Dr. Perper's reliance on pulmonary hypertension and right ventricle hypertrophy to diagnose cor pulmonale. Dr. Hurwitz agreed that the miner had pulmonary hypertension, but he disagreed as to its etiology. Dr. Hurwitz stated that "the presence of pulmonary hypertension secondary to left ventricular dysfunction produce[s] the same pathophysiologic changes in the right heart that an individual with pulmonary hypertension secondary to intrinsic lung disease manifest[s]." (DX 55, p. 2). Dr. Hurwitz concluded that the miner's pulmonary hypertension was due to left ventricular dysfunction based on the severe disease in the left side of his heart, his huge dilated left ventricle with history of congestive heart failure, and the lack of essentially any coronary circulation. Drs. Bush, Griffin, and Tomashefski concurred with Dr. Hurwitz's opinion, and they noted that the miner did not have primary lung disease, a prerequisite of a diagnosis of cor pulmonale. The clinical records support Dr. Hurwitz's findings that the miner had severe left-sided heart disease and essentially no coronary circulation. Also, the pathologists described the miner's coal workers' pneumoconiosis as mild to moderate and the pathologists that quantified the amount of lung affected found that coal workers' pneumoconiosis destroyed five percent or less of the miner's lungs. I find that the evidence fails to establish that the miner had cor pulmonale. However, whether or not the miner had cor pulmonale is not significant because it is not determinative of whether his death was due to pneumoconiosis.

Dr. Mittal concluded that the miner's coal workers' pneumoconiosis and associated emphysema together caused hypoxemia, which contributed to his fatal cardiac arrhythmia. Dr. Perper concluded that the miner's coal workers' pneumoconiosis caused hypoxemia, which precipitated his fatal cardiac arrhythmia. Dr. Bush concluded that the miner's coal workers' pneumoconiosis was too minimal to cause hypoxemia. Drs. Bush, Fino, and Hurwitz found that the objective medical evidence did not reveal evidence of chronic hypoxemia. A review of the objective medical evidence reveals that only three of the pulmonary function studies produced qualifying values and none of the arterial blood gas tests produced qualifying values. The arterial oxygen saturation measurements taken during the miner's July 10, 2000 catheterization, performed two weeks before his death, produced normal values. I find that the objective medical evidence does not support Drs. Mittal and Perper's opinion that the miner's coal workers' pneumoconiosis caused hypoxemia. Because I find that the miner did not suffer from hypoxemia, I accord little weight to Drs. Mittal and Perper's opinion that hypoxemia due to coal workers' pneumoconiosis caused or contributed to the miner's fatal cardiac arrhythmia. *Minnich v. Pagnotti Enterprises, Inc.*, 9 B.L.R. 1-89, 1-90 n.1 (1986).

The death certificate lists coronary artery disease and chronic obstructive pulmonary disease as the immediate causes of death, and coal workers' pneumoconiosis as a significant condition. The record does not contain a letter or report from Dr. Wallace E. Miller, the physician who signed the death certificate, explaining how coal workers' pneumoconiosis contributed to the miner's death. A death certificate alone is not sufficient to establish that coal workers' pneumoconiosis contributed to the miner's death. *Lango v. Director, OWCP*, 104 F.3d 573 (3d Cir. 1997).

Dr. Perper concluded that coal workers' pneumoconiosis contributed directly to the miner's death. Drs. Bush, Fino, Griffin, Hurwitz, Naeye, and Tomashefski concluded that the miner's death was due to severe coronary atherosclerosis. They found that the miner's coal workers' pneumoconiosis was too minimal to have caused or contributed to his death.

Dr. Perper found that the miner had significant simple coal workers' pneumoconiosis with associated centrilobular emphysema and cor pulmonale, which caused a respiratory impairment requiring treatment with bronchodilators. As discussed above, the medical evidence does not support a finding of cor pulmonale. Also, the medical evidence does not establish that the miner suffered from a respiratory impairment during his lifetime, as a preponderance of the pulmonary function studies and arterial blood gas tests produced normal values. The medical record establishes that the miner was prescribed bronchodilators for many years, but it does not establish that the bronchodilators were for the treatment of a coal-dust induced respiratory impairment.[3] Dr. Perper diagnosed slight to moderate coal workers' pneumoconiosis and slight centrilobular emphysema and he concluded that coal workers' pneumoconiosis was a substantial contributory cause of the miner's death. Dr. Perper stated that his opinion was based on his review of the "profuse" medical documentation. (DX 50, p. 31). Dr. Perper stated that the miner's subjective symptoms, physical examination findings, chest x-ray interpretations, pulmonary function study results, and arterial blood gas test results supported his pathologic diagnoses of coal workers' pneumoconiosis and chronic obstructive pulmonary disease. However, Dr. Perper does not explain how this additional information changed his opinion as to the cause of the miner's death.[4] The fact that the miner had conflicting diagnoses of coal workers' pneumoconiosis and chronic obstructive pulmonary disease over the years does not change the severity of the disease identified on the autopsy slides. Because Dr. Perper failed to reconcile his pathological findings with his conclusion that coal workers' pneumoconiosis contributed to the miner's death, I find that Dr. Perper's opinion is poorly reasoned and thus I accord it little weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987).

Dr. Naeye concluded that the miner's death was due to severe coronary arteriosclerosis. However, Dr. Naeye did not make any microscopic findings that support his conclusion. Also, Dr. Naeye stated that the autopsy slides were not representative of the miner's lungs; Employer

[3] Dr. Awan stated that the bronchodilator medication was prescribed for the miner's chronic obstructive pulmonary disease, which was "probably" due to his coal dust exposure. Dr. Awan acknowledged that the miner was unable to undergo testing due to his cardiac catheterization and coronary bypass surgery. Dr. Awan also stated that he had not reviewed any of the miner's pulmonary function studies. (DX 45). Dr. Fino stated that there was no pulmonary reason to prescribe bronchodilators to the miner because his pulmonary function tests were normal. Dr. Fino stated that some individuals with heart-related shortness of breath are prescribed bronchodilator medications because the inhaler forces the individual to stop his activity, thereby allowing the heart to pump enough blood to alleviate the shortness of breath. (EX 1, pp. 18-20). I find that Dr. Awan's opinion is not reasoned because it is not based on any objective medical testing. In contrast, I find that Dr. Fino's opinion is supported by the objective medical evidence of record, and thus accord Dr. Fino's opinion greater weight than Dr. Awan's opinion.

[4] Dr. Perper concluded that coal workers' pneumoconiosis did not contribute to the miner's death in his first medical report. (DX 8).

stipulated at the hearing that the slides were a representative sample of the miner's lungs. (TR 7). Because Dr. Naeye did not adequately consider the role of coal workers' pneumoconiosis in the miner's death and his conclusion that the miner's death was due to severe coronary arteriosclerosis is not supported by his autopsy findings, I find that Dr. Naeye's opinion is poorly documented and reasoned. Therefore, I accord little weight to Dr. Naeye's opinion.

Drs. Bush, Fino, Griffin, Hurwitz, and Tomashefski also concluded that the miner's death was due to severe coronary arteriosclerosis. Drs. Bush, Griffin, and Tomashefski found massive cardiomegaly, bilateral pleural effusions, chronic passive congestion of the lungs and liver, and biventricular myocardial hypertrophy, and concluded that these findings were evidence of the miner's severe cardiac disease. Drs. Fino and Hurwitz stated that the July 10, 2002 catheterization revealed that the miner was "living on a partially occluded single bypass graft." (DX 40, p. 16; *see also* EX 1, p. 35). Dr. Hurwitz explained that the 2002 catheterization and the cardiomegaly at autopsy demonstrate that the miner had "no functional coronary circulation" and that his death was the result of the natural course of his heart disease. (DX 40, pp. 16, 28). Drs. Bush, Fino, Griffin, Hurwitz, and Tomashefski found that the miner's coal workers' pneumoconiosis did not cause or contribute to the miner's death because there is no causal relationship between coal workers' pneumoconiosis and coronary arteriosclerosis and the coal workers' pneumoconiosis was too minimal in severity to contribute to his death. I find that Drs. Bush, Fino, Griffin, Hurwitz, and Tomashefski's opinions provide a reasoned explanation as to the mechanism of the miner's death. I also find that their opinions are supported by the objective medical evidence. Therefore, I accord great weight to the opinions of Drs. Bush, Fino, Griffin, Hurwitz, and Tomashefski.

After reviewing all of the evidence, I find that Claimant has not established her burden under § 718.205(c). Also, there is no evidence in the record that can invoke the § 718.304 presumption. As the evidence does not show that the miner's death was due to pneumoconiosis, the claim will be denied. In light of this denial, Claimant's counsel is precluded from charging a fee for his professional services.

ORDER

IT IS ORDERED THAT the claim of Helen M. Mucker, surviving spouse of George Mucker, for benefits under the Act is DENIED.

A

DANIEL L. LELAND
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this Decision and Order, by filing a notice of appeal with the ***Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601***. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.